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Clinical Study of Acute Cortical Necrosis: In Tertiary Care Center

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Abstract

Introduction: Renal cortical necrosis (RCN) is characterized by patchy or diffuse ischemic destruction resulting in diminished renal arterial perfusion due to vascular spasm and microvascular injury. In addition, direct endothelial injury particularly in setting of sepsis, eclampsia, haemolytic uremic syndrome (HUS) and snake bite may lead to endovascular thrombosis with subsequent renal ischemia. *Aims:* To study clinical features and laboratory parameters with outcome of acute cortical necrosis. *Materials and methods:* It is a prospective study carried out for a period of 2 years in 50 patients presenting with acute renal failure due to acute cortical necrosis.Age group ranged from 2 years to 65 years. *Results:* During this period the total 650 patients presented with acute renal failure . Out of which the biopsy proven renal cortical necrosis were 50 in number. Out of total 50 cases of biopsy proven acute cortical necrosis leading to ARF constitutes pregnancy related were 35 (70%), septicaemia cases were 6 (12%), post gastroentiritiscases were 3 (6%), HUS cases were 3 (6%), snake bite cases were 2 (4%), due to extensive burns was 1 (2%). *Conclusion:* Acute cortical necrosis is most common in female as the commonest cause is of obstetrical in nature. Management consisted of fluid balance and Dialyzes is life saving procedure during recovery period.

Keywords: Renal cortical necrosis; Dialyzes; Acute renal failure.

Introduction

Acute cortical necrosis is characterized by tissue death throughout the cortex with sparing of medullary portion of the kidney. Renal cortical necrosis incidence is higher in developing countries, ranging from 6-7% of all cases of acute renal failure.[1,2] It is a rare cause of acute renal failure, however, in India incidence is increasing due to pregnancy related causes. It is common in late pregnancy, most frequently after placental abruption and less commonly following prolonged intra uterine death or Pre-eclampsia. Acute cortical necrosis occurs in response to a wide range of insults to kidney. Some cases are obvious such as obstetrical causes, septicaemia and others like snake bites, Electrocution and post GE status.

The pattern of acute necrosis in Tropics is slightly different from that of developed countries. Poor socio-economic conditions, prevalence of tropical infections, snake bite and poor obstetrical care in rural areas exercise a significant influence in determining the pattern of diseases in Tropics.[3]

Although acute cortical necrosis involves the entire renal cortex with resultant irreversible renal failure, incomplete or patchy variety occurs more often, characterized by an initial episode of severe oligoanuria and even anuria, lasting longer than uncomplicated tubular necrosis. This is followed by a variable return of function and a stable period of moderate renal insufficiency which in some cases progresses years latter to end stage renal disease.

Renal cortical necrosis (RCN) is a rare cause of acute renal failure secondary to ischemic necrosis of the renal cortex. The lesions are usually caused by significantly diminished renal arterial perfusion secondary to vascular spasm, microvascular injury, or intravascular coagulation. Renal cortial

necrosis is usually extensive, although focal and localized forms occur. In most cases, the medulla, juxtamedullary cortex, and a thin rim of subcapsular cortex are spared.Although the pathogenesis remains unclear, the presumed initiating factor is intense vasospasm of the small vessels.[4] More prolonged vasospasm can cause necrosis and thrombosis of the distal arterioles and glomeruli, and Renal cortical necrosis ensues. In Haemolytic Uremic Syndrome (HUS) and septic abortion, an additional mechanism involves endotoxin mediated endothelial damage that leads to vascular thrombosis. Renal cortical necrosis in placental abruption may be due to a combination of a hypercoagulable state, endothelial injury, and intravascular thrombosis.Failure to consider the diagnosis in a pregnant woman with sudden onset of abdominal pain, a tender uterus, and haematuria, especially during the third trimester. Failure to consider the diagnosis in a new born child with dehydration, oliguria, and haematuria may lead to legal consultation.

Materials and Methods

This study was carried out in the Department of Nephrology, during the period August 2016 to July 2018 in collaboration with Department of Histopathology. It is a prospective study of 50 patients presenting with acute renal failure due to acute cortical necrosis.

Inclusion criteria: Presence of clinical manifestations of acute renal failure i.e., Serum creatinine > 2 mg/dl and raised renal parameters at admission, Biopsy proven histological varieties of all cases of ARF with acute cortical necrosis.

Exclusion criteria:

Presence of chronic renal failure.

Obstetrical-cases presenting with abruption placenta and prolonged labour with septicaemia.

Septic ATN-Criteria mentioned by Liano et al was used i.e.ATN occurring in presence of bacteraemia or a known focus of infection with documentation of two of the following: They are rigors, unexplained hyperventilation, unexplained fall in blood pressure, abrupt rise in temperature more than 38 degrees C not due to transfusion reaction or unexplained leucocytosis of more than 15,000/ cumm.

Snake bite with slowly developing ARF with or without septicaemia.

Gastro-enteritis with history of diarrhoea and vomiting.

Burns associated with acute renal failure.

A detailed history and clinical profile of these patients were recorded as per the proforma. The duration of the incidence and the time period elapsed between onset of the disease and development of renal failure were recorded.

Blood urea, serum creatinine and electrolytes (sodium, potassium, chloride) were done daily and recorded. Other laboratory parameters such as blood glucose, complete urinary examination, hemoglobin, total leucocyte count and differential count, erythrocyte sedimentation rate at the end of one hour, platelet count were done at admission and recorded. Additional investigations performed were, prothrombin time, liver function tests, (serum bilirubin, total serum protein, serum albumin, alkaline phosphatise, transaminases,) hemoglobinuria, myoglobin, urinary protein electrophoresis in certain cases. GE and development of renal failure were recorded. The hydration status at time of admission was recorded. Blood urea, serum creatinine and electrolytes (sodium and potassium) were done daily and recorded. Other laboratory parameters such as blood glucose, complete urinary examination, hemoglobin, total leukocyte count and differential count, erythrocyte sedimentation rate at end of one hour, platelet count, were done at admission and recorded. Additional investigations performed were, prothrombin time, liver function test (serum bilirub in, total serum protein, serum albumin, alkaline phosphatise, transaminases).

These patients were given adequate fluid replacement based on severity of dehydration as a first step in management. The complications such as hyperkalaemia and metabolic acidosis were corrected on emergency basis. Daily input and output fluids chart were maintained and fluids were given accordingly. Antibiotics which have gram negative spectrum preferably quinolones were started to patients with ongoing gastroenteritis. Change of antibiotics was done whenever deemed necessary based on the doubt of complications or reports of culture sensitivity. Dialyzes was done in patients with hyperkalaemia, pulmonary oedema

and severe metabolic acidosis, who did not respond to medical treatment and prophylactically in patients whose creatinine is more than 8 mg/dl. All patients were followed up till discharge or death and complications that occurred in their hospital stay were recorded. The clinical and laboratory parameters were analysed to assess the role of each of these factors as the possible outcome i.e., recovery or death.

Results

The study was carried out between August 2006 to July 2008. During this period the total number of patients presented with acute renal failure (In acute medical care unit, medical ward, obstetric units and Nephrology units) were nearly 650. Out of which the biopsy proven renal cortical necrosis were 50 in number. Out of total 50 cases of biopsy proven acute cortical necrosis leading to ARF constitutes pregnancy related were 35 (70%), septicaemia cases were 6 (12%), post-G.E.cases were 3 (6%), HUS cases were 3 (6%), snake bite cases were 2 (4%), due to extensive burns was 1 (2%).Out of 50 patients, the age group ranged from 2 years to 65 years.

Baseline creatinine at the time of admission ranged between 2.2 to 20mg/dl. The mean peak creatinine was 4-9mg/dl in survivors, and 5-8 mg/dl in non-survivors.

Table 1: Demographic Distribution in study

Age Group	Female	Male	Total (%)
0-10		1	2%
11-20	8	1	18%
21-30	13	–	26%
31-40	16	7	46%
41-50	1	2	6%
51-60	–	–	Nil
>60	–	1	2%

Out of 50 Patients 38 (76%) were females and 12 (24%) were males.

Table 2: Clinical presentation in present study

Clinical features	Number of cases	Percentages
Azotemia	50 cases	100%
Oligo-anuria	40 cases	80%
Volume overload	30 cases	60%
Respiratory Distress	25 cases	50%
HTN	20 cases	40%
Haematuria	10 cases	20%
Hypotension	8 cases	16%
Sepsis	6 cases	12%

All the cases in our study have azotemia (100%)followed by oligo-anuria(80%)

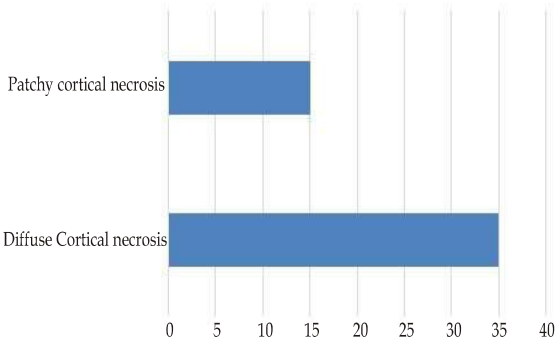

Fig. 1: Histological variety in present study
Diffuse cortical necrosis is most common necrosis observed in study 35 cases(70%)

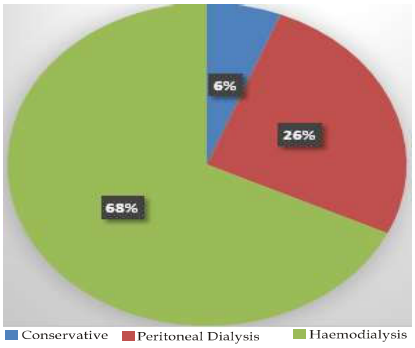

Fig. 2: Treatment Schedule in present study.
68% of patients in present study treated with haemodialyzes , 26% cases treated by peritoneal dialyzes.

At admission the serum urea levels ranged between 49 to 330 mg/dl with more number of patients presenting between an average of 100-200mg/dl, whereas at the time of discharge the range is between 24mg/dl to 241 mg/dl with majority of patients averaging between 40 to 80mg/dl.

Sixteen patients had hyponatremia (<125 mEq/dl) at the time of admission. During hospital course 8 more patients developed hyponatremia. Overall 24 (47.03%) patients had a hyponatremia.18 patients had hypernatremia (>150 mEq/dl)

Serum potassium ranged between 2 mEq/dl to 7.2 mEq/dl,42 (82.3%) patients presented with hypokalaemia. Out of them 21 (41.1%) presented at the time of admission. Hyperkalaemia occurred in 22 (42%) patients and 14 out of them presented at the time of admission. All non-survivors had hypokalaemia.

Hypophosphatemia (<3mg/dl) was observed in 4 patients and all of them survived. Hyperphosphatemia (>5mg/dl) occurred in 17 patients out of them 2 died.

Hypocalcaemia (<8mg/dl) occurred in 7 (13.7%). Hypercalcemia occurred in 2 (3.9%) patients. Disseminated intravascular coagulation 10 (19.6%) had prolonged PT and they were considered to have DIC. Out of them 4 died and 6 survived. Anaemia (Hb < 10gm%) was seen in 14 patients and all of them survived.None of patients were positive for Hbs Ag or HIV.Pneumonia was seen in 6 (11.7%) patients, pulmonary edema in 2 (3.92%) and one of the died. Pleural effusion was observed in 3 (5.8%), ARDS occurred in one patient. No patient had organomegaly, ascites or abnormality in kidneys.

Out of 50 patients 37 (74%) survived and 12 (26%) died.13 patients under gone P.D. 34 patients undergone H.D and 3 patients maintained conservatively. Most of the patients who died were due to Septicaemia and multi-organ failure.

Discussion

The study was conducted over a period of two years from August,2016 to July 2018. Out of 650 cases of acute renal failure studied, 50 cases were of renal cortical necrosis. All the cases were subjected to renal biopsy. Renal cortical necrosis constitutes 7.6% of all cases of acute renal failure. Pregnancy related cases were 35 (70%), septicaemia cases were 6 (12%), Post GE cases were 3 (6%), HUS were 3 (6%), Snake bite cases were 2 (4%) and due to burns was 1 (2%).

Renal cortical necrosis was responsible for 3.8% patients dialysed for acute renal failure at a centre in North India studied by Chugh K.S.et al at Department of Nephrology, Post graduate Institute of Medical Education and Research, Chandigarh. 5 In our study the renal cortical necrosis is 50 out of 650 cases which comes to 7.6%.In contrary to studies made by Chugh K.S.et al, the ratio is high and the incidence is slowly increasing because of early referral to Tertiary referral centres and the early interventions made to diagnose the cases and giving a proper treatment to save the patient which would have died because of acute renal failure which is not exactly classified into the category and not explored to renal biopsy.But, this ratio of 7.6% corresponds well with the studies made by Prakash. J et al at Institute of Medical Sciences, Banaras Hindu University, Varanasi.1 They report a 23 cases of biopsy proven renal cortical necrosis which constituted 6.3% of all cases of acute renal failure. This study correlates with our study which shows 7.6% cases of renal cortical necrosis. The patients were divided into two groups : Obstetric and non-obstetric. Obstetric complications were responsible for renal cortical necrosis in 15 (65%) patients while non-obstetric conditions accounted for the remaining 8 (35%) cases. This corresponds to our study in which out of 50 cases 35 (70%) are related to obstetric origin and 15 (30%) cases belong to non-obstetric origin.Haemolytic Uraemic Syndrome 3(12%) cases and septicaemia 2 (8%) cases were the main causes of necrosis in the non-obstetric group. This corresponds to our study which shows that this non-obstetric group constitutes to 18% of all cases. The cortical necrosis pathologically was divided into two groups. One is diffuse cortical necrosis and the other is patchy cortical necrosis. Prakash. J et al conformed in the study that cortical necrosis was diffuse and patchy in 17 and 6 patients respectively which is calculated to 70% and 30% in the two pathological verities. Out study shows diffuse cortical necrosis in 65% cases and patchy cortical necrosis in 35% cases which is nearer to Prakash.J.et al studies.[1,6] The disease had a fatal prognosis in 20 (87%) patients. Mortality was due to uraemic complications and infections in the majority of patients. In our study till the discharge the patients were on dialyzes and most of the people not turned up to the institution. Out of 50 cases 13 (26%) died and other 37 (74%) were discharged at request and not turned up to

institution. So, we do not know the prognosis or mortality rate of that group of patients. Most of the patients who died were due to septicaemia and multi-organ failure. Within the obstetric causes of cortical necrosis Chug KS et al confirmed by his study that septic abortions were 27 (42%) cases and other late pregnancy related complications were 36 (58%) cases.[5] In our study we never come across any case with abortion related renal cortical necrosis. This is true that now a days most of the people are adopting family planning programmes and visiting health care centres at an early date to avoid complications. In our study we have not found any renal cortical necrosis case with misuse of drugs or complicated by routine drugs or poisons. Prakash J et al noticed in their study that renal cortical necrosis due to haemolytic uraemic syndrome in observed in 16 (39%) children and majority (76%) of the cases had a diarrheal prodrome.[6] In our study no child is admitted with diarrhoea induced cortical necrosis because they are usually referred and treated in paediatric institutes.

Renal cortical necrosis accounts for 2% of all cases of acute renal failure in adults and 15-20% of ARF during the third trimester of pregnancy in developed nations. However, renal cortical necrosis incidence is higher in developing countries ranging from 6-7% of all cases of acute renal failure. In contrary, to the incidence rate seen in developed countries our study is consistent with the incidence rate of renal cortical necrosis which is seen in developing countries.

The causes of death during acute phase of illness are; severe uraemia, sepsis, pulmonary oedema, gastrointestinal haemorrhage and hyperkalemia including multiorgan failure.[7] Thus, the majority of deaths are due to sepsis and uremic complication in those who could not afford dialyzes. However, the prognosis and survival of patients with RCN has improved markedly in our recent publication due to availability of renal replacement therapy and overall improved medical care.[8,9] Survival without dialyzes is possible in patients with patchy cortical necrosis because surviving nephrons carry the function of the remaining kidney. In certain patients, there may be slow rise in creatinine clearance and a gradual gain in renal function over one to two years, so that the glomerular filtration rate may reach a final plateau level of approximately 20-24 mL/min. It is assumed that juxtamedullary glomeruli (which comprise 15%-20% of total) escape destruction, even in the complete cortical necrosis and that early functional return is due to recovery of these nephron segment. The deterioration in renal function had been reported several years (1-10 years) after the acute cortical necrosis in a significant number of patients. Factors causing these late functional downturn are not clear but may include pyelonephritis, hypertension and shrinkage of the kidney due to progressive fibrosis and/or calcification.[10]

Conclusions

Acute cortical necrosis is one of the rate causes of acute renal failure.Most of the etiological factors of acute cortical necrosis is of obstetrical in nature.The prognosis of acute cortical necrosis is worst than other types of acute renal failure. Acute cortical necrosis is most common in female as the commonest cause is of obstetrical in nature.Management consisted of fluid balance and Dialyzes is life saving procedure during recovery period.Though acute cortical necrosis was rarest cause of ARF, now the incidence is slowly increasing.

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